ENVIRONMENTAL PROTECTION AGENCY

[OPTS-4207; BHTSH FRL 2443-1]

Ethylene Oxide; Response to the Interagency Testing Committee

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The First Report of the Interagency Testing Committee (ITC), transmitted to EPA in October 1977, designated the category of alkyl epoxides for consideration by EPA for health and environmental fate testing. This Notice provides EPA's response to the ITC's recommendations with respect to ethylene oxide, one member of the alkyl epoxides category. Other category members will be addressed in separate Federal Register notices. In view of the accumulating data base and the current regulatory activities underway on ethylene oxide by the Occupational Safety and Health Administration (OSHA) and EPA's Office of Pesticide Programs (OPP), EPA has concluded that additional health effects testing of ethylene oxide should be pursued by EPA only if OSHA or OPP concludes that such additional testing is necessary and requests support in gathering test data under the Toxic Substances Act (TSCA). EPA believes that existing data are adequate to reasonably predict the environmental fate of ethylene oxide. Consequently, EPA is not initiating rulemaking under section 4(a) of TSCA to require health or environmental fate testing of ethylene oxide at this time.

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SUPPLEMENTARY INFORMATION:

I. Background

Section 4(a) of the Toxic Substances Control Act (TSCA) (Pub. L. 94-469, 90 Stat. 2003 et seq.; 15 U.S.C. 2601 et seq.) authorizes EPA to promulgate regulations requiring testing of chemical substances and mixtures to develop data relevant to assessing the risks that such chemicals may present to health and the environment.

Section 4(e) of TSCA established an Interagency Testing Committee (ITC) to recommend to EPA a list of chemicals to be considered for the promulgation of testing rules under section 4(a) of TSCA. The ITC placed the alkyl epoxides category on its first priority testing list published in the Federal Register of October 1977 (42 FR 55026). The ITC recommended that testing be considered for the alkyl epoxides for carcinogenicity, mutagenicity, teratogenicity, other chronic effects, and environmental; effects. The ITC recommended that the chronic effects testing consider organ effects and behavioral changes and that the environmental testing focus on the fate of epoxides in the environment. Epidemiological studies were also recommended for two or three of the highest exposure compounds if suitable cohorts could be identified.

The alkyl epoxides category, as defined by the ITC, includes all noncyclic aliphatic hydrocarbons with one or more epoxide functional groups. This notice addresses a single member of this category, ethylene oxide. Other members of the category will be addressed in other Federal Register notices.

Approximately 5 to 6 billion pounds of ethylene oxide is produced annually in the United States. Over 99 percent of the ethylene oxide produced is consumed as a chemical intermediate. Less than 1 percent of the ethylene oxide produced is used as a sterilant or fumigant. However, these latter uses are regulated by EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and by the Food and Drug Administration (FDA), and therefore releases to the environment and exposures from these uses have not been considered in this notice. EPA's responses to the ITC's specific recommendations are set forth below with respect to ethylene oxide.

II. Analysis of the ITC's Concerns

A. Carcinogenicity

A chronic inhalation bioassay with ethylene oxide has been completed at Union Carbide's Bushy Run Research Center. Exposure levels were 10, 33, and 100 parts per million (ppm). A statistically significant increase in mononuclear cell leukemia was found in female rats exposed to 100 ppm. In addition, increased incidences of mononuclear cell leukemia for the females exposed at 33 and 10 ppm, although not statistically significant over controls, suggest a dose-response relationship. An increase in peritoneal mesothelioma was reported in the male rats exposed at 33 and 100 ppm. Among the males exposed at 100 ppm, the cumulative percentage developing a tumor of this type was reported to be statistically significantly higher than that [illegible]e controls beginning with the 21[illegible]th of exposure. The incidence of [illegible]se tumors in males exposed at 33 p[illegible] was not appreciably higher than in [illegible]e controls until the final month of the study. These peritoneal tumors originated in the testicular mesothelium and were confined to the abdominal cavity (Ref. 1). A statistically significant increased incidence of primary brain neoplasms in the male rats exposed to 100 ppm ethylene oxide and an increased incidence (not statistically significant) of primary brain neoplasms for males exposed to 33 ppm and for females exposed to both 100 and 33 ppm ethylene oxide was subsequently reported (Ref. 2).

Preliminary results from a two-year chronic inhalation study conducted by the National Institute for Occupational Safety and Health (NIOSH) on male rats and male monkeys were reported at the 1982 meeting of the Society of Toxicology (Ref. 3). In that study, groups of 80 male Fischer 344 rats and 12 male Cynomolgus monkeys were exposed to 50 ppm and 100 ppm ethylene oxide over a two year period. Two groups, 80 rats and 12 monkeys, were used as controls and exposed to conditioned, filtered ambient air. During the study, all of the rat groups became infected with *Mycoplasma pulmonis* which, beginning with the sixteenth month, caused the death of a large segment of the rat population. The preliminary results of the available histopathological evaluation of the spleen indicated an exposure-related increase of mononuclear cell leukemia in male rats exposed to ethylene oxide at 50 ppm but not at 100 ppm. NIOSH has acknowledged that these preliminary results must be interpreted in light of the known spontaneous incidence of leukemia in Fischer 344 rats, but notes that excess mortality has occurred in the 100 ppm group (19% survived as compared to 49% of the controls). At the terminal kill, a significantly higher frequency of leukemia was found only in the group exposed to 100 ppm of ethylene oxide. Of equal or greater importance, however, is the apparent dose-related finding of gliomas in the rats of the NIOSH study. This tumor is rare in Fischer 344 rats. Gliomas were found in 5 of 79 rats exposed at 100 ppm and 2 of 77 rats exposed at 50 ppm. There were none in the 76 control rats. A significant association of exposure and an occurrence of peritoneal mesothelioma was found for rats exposed to 100 ppm ethylene oxide, but not to 50 ppm ethylene oxide. These results parallel those from the Bushy Run study. None of the monkeys in the

NIOSH study have demonstrated any evidence of leukemia to date but they are still being monitored. A more comprehensive evaluation of the chronic studies is planned by NIOSH after further data analysis. In addition, the National Toxicology Program (NTP) is sponsoring a chronic bioassay via inhalation with ethylene oxide in mice. The exposure phase of this study was completed in July, 1983.

Available data and the data to be obtained from these ongoing studies are sufficient to reasonably determine the carcinogenicity of ethylene oxide. The current OSHA and OPP regulatory actions on ethylene oxide are based in part on the excess risks to humans presented due to carcinogenicity.

B. Mutagenicity

Ethylene oxide gave positive results in gene mutation assays in: (1) prokaryotes (Refs. 4, 5); (2) eukaryotes (Refs. 6–13); (3) the *Drosophila* sex-linked recessive lethal mutation assay (Refs. 14, 15); and (4) the *Drosophila* autosomal deletion mutation assay (Refs. 15). There is an ongoing evaluation of the mutagenicity testing sponsored by EPA and conducted at Oak Ridge National Laboratory (ORNL) and Louisiana State University on: (1) alkylation in *Drosophila* sperm cells, (2) alkylation in mouse sperm cells, and (3) mouse specific locus test. In addition, the National Institute of Environmental Health Sciences (NIEHS) is conducting a biochemical specific locus assay in mice.

Positive results were obtained in the following tests to detect chromosomal aberrations: (1) dominant lethal in mice (Refs. 16–18); (2) dominant lethal in rats (Ref. 19); (3) micronucleus test (Ref. 20); (4) heritable translocation in mice following intraperitoneal injection (Ref. 17); (5) chromosomal abnormalities in rat bone marrow cells (Refs. 4, 21–23); and (6) chromosomal aberrations in the cultured lymphocytes of Cynomolgus monkeys (Ref. 3). In addition, a heritable translocation test in mice inhalation sponsored by NTP is in progress at ORNL. The NTP protocol calls for exposure concentrations of 50, 100, 150, 200, and 255 ppm.

Positive results were obtained in the following studies to detect primary DNA damage; (1) unscheduled DNA synthesis in mice (Ref. 24); and (2) increases in sister chromatid exchanges (SCE's) in cultured lymphocytes of Cynomolgus monkeys exposed to ethylene oxide via inhalation (Ref. 3).

Indications of chromosomal changes in humans resulting from occupational exposure to ethylene oxide have also been reported. These include increases in chromosomal aberrations (Refs. 25, 26) and sister chromatid exchanges (Refs. 26–29) in human lymphocytes.

Available data and data from ongoing mutagenicity studies are sufficient to reasonably determine the mutagenic effects of ethylene oxide. The current OSHA and OPP regulatory actions are based partially upon the evidence of mutagenicity for ethylene oxide.

C. Teratogenicity

A discussion of the data from teratogenicity testing of ethylene oxide in the mouse, the rat, and the rabbit follows.

Mouse. La Borde and Kimmel (Ref. 30) reported the results of a teratogenicity study in the CD–1 mouse following intravenous injections of ethylene oxide in 5% dextrose at doses of 75 and 150 mg/kg. Four groups of mice were treated daily for 3 days at each of the following periods of gestation: days 4–6 (Period I), 6–8 (Period II), 8–10 (Period III), and 10–12 (Period IV). Cervical and thoracic skeletal abnormalities were noted in fetuses in the 150 mg/kg group exposed during period II. This dose also produced severe effects in the dams exposed during periods I, III, or IV but not during period II. A dose-response relationship was not evident in any of the periods; the incidence of effects (both maternal and fetal) was similar for animals treated at 75 mg/kg and for controls.

Rat. Results of an inhalation teratology study sponsored by NIOSH in Sprague-Dawley CD rats have been reported (Ref. 31). Maternal toxicity, reproductive performance, and developmental toxicology were evaluated following 7 hr/day inhalation exposures to 150 ppm ethylene oxide. Rat exposure regimens were: (1) filtered air (control); (2) chemical exposure from days of gestation (dg) 7 through 16; (3) chemical exposure from dg 1 through 16 and (4) chemical exposure for 5 days/wk for 3 weeks prior to mating and daily from dg 1 through 16. Unexposed males were used in mating.

Reduction in food consumption and body weight were significant in rats exposed before breeding and rats exposed dg 1 through 16. The incidence of resorptions was significantly increased only in litters from rats exposed before breeding. Fetal weight and crown-rump length were reduced in litters from all ethylene oxide-exposed groups of rats. Fetal morphologic changes included reduced ossification of the skull and sternebrae in litters from all ethylene-oxide-exposed groups and an increased incidence of hydroureter (not statistically significant) in litters exposed from dg 7 through 16.

Significant adverse effects in development were observed in the group exposed dg 7 through 16 in the absence of any significant adverse effects on maternal body weight gain or food consumption.

Results from another inhalation teratology study in rats at the Bushy Run Research Center have also been reported (Ref. 32). Pregnant Fischer 344 rats were exposed 6 hours daily to 10, 33, or 100 ppm ethylene oxide on dg 6 through 15. No treatment-related effects were noted in the dams. Fetal weights for both males and females were significantly depressed, and an increased frequency (not statistically significant) of delay ossification was noted in the 100 ppm group. No effects from exposure were noted for the dams or fetuses in the 33 and 10 ppm groups.

Rabbit. Intravenous studies were carried out by Kimmel et al. (Ref. 33) in rabbits at doses of 0, 9, 18, and 36 mg/kg administered intravenously daily on dg 6 through 14 or doses of 0, 18, and 36 mg/kg daily on dg 6 through 9. Preliminary studies had indicated the maximum tolerated dose (MTD) to be approximately 40 mg/kg. A statistically significant trend toward decreased maternal weight gain with increasing dose was seen during treatment and throughout gestation after treatment either on dg 6 through 9 or 6 through 14. No significant effects were seen in the fetal parameters examined after treatment on dg 6 through 9. However, a significant increase in mean number and percent resorptions/litter was noted in the 36 mg/kg dose group treated on dg 6 through 14. Thus, ethylene oxide administered intravenously to pregnant rabbits increases the incidence of adverse effects on development, after treatment throughout organogenesis at a dose that also produces maternal toxicity. Unlike the effect of ethylene oxide in the mouse following intravenous administration, no structural malformations were detected in rabbits in this study.

Results of an inhalation teratology study sponsored by NIOSH in New Zealand white rabbits have also been reported (Ref. 31). Rabbits were artificially inseminated and placed on one of the following exposure regimens at 150 ppm for 7 hours/day: (1) filtered air (control); (2) chemical exposure from dg 7 through 19; and (3) chemical exposure from dg 1 through 19. No evidence of maternal toxicity, adverse effects on development or structural malformations was detected in rabbits exposed to 150 ppm of ethylene oxide.

EPA concludes that the data from the above studies are sufficient to

reasonably determine the teratogenicity of ethylene oxide.

D. Other Chronic Effects

As a matter of general policy under section 4 of TSCA, EPA generally accepts data from well-conducted oncogenicity studies as being sufficient to assess the chronic toxicity of a chemical. EPA has concluded that adequate data are and will be available, from the completed oncogenicity studies, from the ongoing oncogenicity studies, and from the various subchronic studies which have been conducted, to reasonably determine the other chronic effects of ethylene oxide, except for reproductive and neurotoxic effects.

E. Reproductive Effects

The final report of a one-generation reproductive study in rats by the Bushy Run Research Center is available (Ref. 34). Both male and female rats were exposed to doses of 10, 33, or 100 ppm of ethylene oxide vapor. Statistically significant observations in the 100-ppm exposure group were decreased implantations, smaller litters, and increased length of gestational period. No treatment-related effects were noted in either the dams exposed to 33 or 10 ppm ethylene oxide or in their litters. Because this study was only a one-generation study, EPA does not believe that it was fully adequate to assess the reproductive effects of ethylene oxide.

Nevertheless, as discussed in Unit III of this notice, EPA has concluded that, in view of the ongoing regulatory activities on ethylene oxide by OPP and OSHA, additional reproductive effects testing of ethylene oxide should only be required under TSCA if requested by OSHA or OPP in support of their regulatory efforts.

F. Neurotoxicity

Paralysis, muscular atrophy of the hind limbs, and growth depression were observed in subchronic studies in rats, rabbits, and monkeys exposed to 357 and 204 ppm ethylene oxide vapor (Ref. 35). Peripheral neuropathy was reported in four workers who were accidentally exposed to high levels of ethylene oxide over a two-month period at a plant where hospital products were sterilized (Ref. 36). The NIOSH chronic inhalation bioassay (Ref. 3), discussed in Unit II.A. of this notice, includes an evaluation of neuropathology and neurophysiology in Cynomolgus monkeys. Exposure levels in the NIOSH chronic bioassay were 50 and 100 ppm via inhalation over a two-year period. The results of the neuropathological evaluation have recently been reported (Ref. 37). Two of the twelve monkeys in each exposure group were sacrificed for neuropathological evaluation. The only significant finding was an increase in axonal dystrophy in the nucleus gracilis of the experimental monkeys as compared to the two controls and demyelination of portions of the gracile tract in one of the monkeys in each of the low and high dose groups.

As discussed in Unit III of this notice, in view of the ongoing regulatory activities on ethylene oxide by OPP and OSHA, EPA has concluded that additional Neurotoxicity testing of ethylene oxide, including testing for behavioral changes, should be pursued under TSCA only if requested by OSHA or by OPP to support their ongoing regulatory activities.

G. Epidemiology

As a consequence of three observed cases, Hogstedt et al. (Ref. 38) reported an apparent excess of leukemia among Swedish workers in a factory where a mixture of ethylene oxide and methyl formate had been used to sterilize hospital equipment. In another study, Hogstedt et al. (Ref. 39) reported the results of a historical prospective mortality and cancer morbidity investigation of 89 workers in an ethylene oxide production facility. These workers may also have been exposed to other chemicals. Among 23 deaths, 9 cancer deaths were observed compared with 3.4 that were expected. The significance of the above epidemiological findings is limited by the small number of observed deaths, the uncertainty of worker exposure information, and the inability to attribute the observed mortality to a particular chemical. Morgan et al. (Ref. 40) reported on a mortality study cohort of 787 production workers potentially exposed to ethylene oxide. Industrial hygiene measurements reportedly revealed no detectable ethylene oxide levels in the product area. At the sources of ethylene oxide (pumps, valves, pipe flanges, spigots, and gauges), less than 10 ppm was recorded. Only during tank car loading operations were levels of approximately 6,000 ppm ethylene oxide recorded. All other measurements were below 50 ppm. The researchers saw fewer than expected deaths from all causes and fewer than expected deaths from total malignancies. The standardized mortality ratios were 58 to 79, respectively. No death from leukemia was observed as compared to 0.70 expected. There were, however, a total of 8 deaths reported for pancreatic cancer, bladder cancer, brain and CNS cancer and Hodgkin's disease compared to 2.16 expected for this worker group.

In addition, at least two mortality studies are in progress. NIOSH is conducting a study on occupational exposure to ethylne oxide at a Union Carbide plant in West Virginia. EPA's Office of Research and Development (ORD) is funding an epidemiological study at Columbia University of hospital workers exposed to ethylene oxide.

Recently, Hemminki et al. (Ref. 41) reported an increase in the number of spontaneous abortions of Finnish hospital workers exposed to ethylene oxide. From other studies, the authors inferred the 8-hour time weighted average (TWA) to be 0.1 to 0.5 ppm with peak concentrations up to 250 ppm. Due to certain methodological problems, this study does not sufficiently define the effects of ethylene oxide exposure on female reproduction. The finding of spontaneous abortions in ethylene oxide-exposed humans, however, does raise questions about reproductive effects. Further efforts may be needed to address this issue. Because of the small number of women exposed to ethylene oxide during its manufacture (Ref. 42), confirmatory epidemiological studies would have to be carried out on hospital workers, where the use of ethylene oxide is not covered by TSCA. A reproductive outcome study of workers potentially exposed to ethylene oxide in hospitals, partially funded by the March of Dimes, has recently begun at the State University of New York at Buffalo. NIOSH has also expressed an interest in performing an epidemiological study of the effects of ethylene oxide on males. In addition, OSHA's proposed rule on ethylene oxide (Ref. 43) includes workplace exposure monitoring and a requirement for worker medical surveillance.

As discussed in Unit III of this notice, in view of the completed and ongoing epidemiological efforts on ethylene oxide, EPA has concluded that a requirement for additional epidemiological studies on ethylene oxide under TSCA does not appear necessary at this time. The TSCA authority could be utilized at a later time if the ongoing epidemiological activities prove to be inadequate and if such additional work is considered necessary to support OSHA or OPP regulatory activities.

H. Environmental Fate

The ITC expressed concern for the reaction products of alkyl epoxides in the environment. Therefore, it recommended that the fate of epoxides in the environment should be determined through testing. EPA has concluded, however, that there are

sufficient data to reasonably predict the environmental fate, including the characterization of degradation products, of the ethylene oxide that might be released during manufacture, distribution in commerce, processing, use, and disposal, and that there is no need for EPA to require testing to better characterize the fate of such releases.

Ethylene oxide is produced by the direct oxidation of ethylene. Almost 90 percent of the ethylene oxide produced is used by its manufacturers as an intermediate or a raw material for the manufacture of other products. Over 9 percent is sold to other firms for similar use (Ref. 44). As explained earlier, sterilant or fumigant uses are regulated under FIFRA and by FDA; therefore, environmental releases from these uses have not been considered in this notice.

Ethylene oxide is manufactured, processed, and distributed in systems engineered to prevent escape of ethylene oxide to the surrounding air. In a letter from the Ethylene Oxide Industry Council (EOIC) to EPA dated December 7, 1981, estimates of the release of ethylene oxide to the atmosphere were submitted. According to the EOIC, the primary source of environmental exposure is through release into the air. The EOIC estimates that about 3 million lbs/year is released to the air (Ref. 45). In a report prepared by Science Applications, Inc. (SAI) for EPA, total nationwide atmospheric emissions of ethylene oxide in 1978 from all sources were estimated to be 1,991,000 lbs (Ref. 46). Results from an atmospheric dispersion model predict average annual exposure levels of ethylene oxide near production plants to be very low, i.e., < 10 ug/m^3 (< 6 ppb) (Ref. 46). In addition, the chemistry of ethylene oxide is such that it will be hydrolyzed by water vapor and oxidized by hydroxyl free radicals in the atmosphere. An anticipated atmospheric degradation product is formic anhydride, OHCOCHO, which reacts with water to give formic acid (Ref. 47).

In its December 7, 1981, letter the EOIC also stated that the amount of ethylene oxide lost to water during production and processing was 800,000 lbs. annually; however, most producers reported to the EOIC that this waste water containing ethylene oxide is treated in a biopond before being discharged from the plant (Ref. 45). Ethylene oxide reacts readily with water to form ethylene glycol. The hydrolysis half-life of ethylene oxide in river water at 25° C and pH 7.4 was 14.2 days (Ref. 48). In sterile river water and sterile distilled water, the hydrolysis half-lives were 12.9 and 12.2 days, respectively (Ref. 48). The epoxide functional group of ethylene oxide readily reacts with other nucleophiles, such as the chloride ion, by pathways that parallel hydrolysis. The hydrolysis/hydrochlorination half-life of ethylene oxide in salt water is about 9 days at 25° C (Ref. 48). The ratio of chlorohydrin to glycol formed is about 0.2 at 3% NaCl; the ratio is directly proportional to salt concentration (Ref. 48). Biodegradation within the water column should further decrease ethylene oxide concentrations. In three BOD tests without prior acclimation, ethylene oxide was biodegraded 75, 69, and 52 percent in 20 days (Refs. 48–50). The desorption rate of ethylene oxide from natural waters was estimated to be 0.36 times that of oxygen under the same conditions (Ref. 48).

On the basis of the environmental release, waste treatment, and environmental fate information on ethylene oxide discussed above, EPA concludes that sufficient data exist to reasonably predict the environmental fate, including the characterization of degradation products, of the ethylene oxide that might be released during the manufacture, distribution in commerce, processing, use, and disposal of ethylene oxide and that there is no need for EPA to require testing to better characterize the fate of such releases.

III. Decision Not To Initiate Rulemaking

EPA has decided not to initiate rulemaking at this time under section 4(a) of TSCA to require further health and environmental fate testing of ethylene oxide. This decision is based on a review of the available data and ongoing testing for this chemical and on regulatory actions being undertaken by EPA and OSHA.

OSHA has recently published a proposed rule which would lower the permitted exposure limit (PEL) of ethylene oxide to an 8 hour time-weighted average (TWA) of 1 ppm. The proposal would provide for certain methods of exposure control, personal protective equipment, measurement of employee exposures, training, medical surveillance, signs and labels, regulated areas, emergency procedures and record keeping, among other requirements (Ref. 43). OPP has previously issued a Rebuttable Presumption Against Registration (RPAR) (Ref. 51) against ethylene oxide and plans in the near future to issue a proposal in the Federal Register to change pesticide labels and adopt other appropriate measures which will help to lower exposure levels of ethylene oxide resulting from its use as a pesticide.

EPA has sufficient data to reasonably predict the environmental fate of ethylene oxide, and therefore EPA concludes additional environmental fate testing is unwarranted. In view of the ongoing epidemiological efforts on ethylene oxide as discussed in Unit II. G. of this notice, EPA concludes that a requirement for additional epidemiological studies on ethylene oxide under TSCA does not appear necessary at this time. The most recent information available to EPA indicates that available data and ongoing testing are adequate to characterize ethylene oxide's carcinogenicity, mutagenicity, teratogenicity, and other chronic effects except reproductive and neurotoxic effects (including behavioral changes). Although the available health effects data may not be sufficient to thoroughly assess the neurotoxicity or reproductive effects of ethylene oxide, the Agency is not initiating rulemaking to require additional testing for these effects at this time for a number of reasons. First, the available data indicate that ethylene oxide is a carcinogen and may produce other health hazards. The Agency believes that the available data on carcinogenicity and other effects are sufficient to support regulatory action to control exposure from uses governed by TSCA. As noted above, OSHA already has proposed to lower the occupational exposure level for ethylene oxide to a PEL of 1 ppm. Moreover, the occupational exposures subject to OSHA's proposal include the exposures that would be subject to the Agency's authority under TSCA.

In addition, the Agency believes that the 1 ppm TWA level proposed by OSHA will reduce significantly not only the cancer risk from worker exposure to ethylene oxide but will also substantially reduce the potential risks from other health hazards including any neurotoxic and adverse reproductive effects. The Agency believes that additional testing does not need to be conducted in order to adequately support the 1 ppm TWA exposure level proposed by OSHA or even a lower exposure level. Thus, the Agency concludes that no significant additional benefit to society will result from requiring further testing for neurotoxicity or reproductive effects at this time. OSHA and OPP are aware of EPA's activities on ethylene oxide under section 4 of TSCA. If either entity should determine at a later time that additional testing of ethylene oxide is necessary, EPA will consider requiring additional testing under TSCA section 4.

IV. References

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V. Public Record

The EPA has established a public record for this testing decision (docket number OPTS–42027). This record includes:

1. Federal Register notice designating the alkyl epoxides category to the priority list and all public comments on ethylene oxide received in response to that notice.

2. Letters.

3. Contact reports of telephone conversations and meeting summaries.

4. Published and unpublished data.

This record, containing the basic information considered by the Agency in developing this decision, is available for inspection in the Office of Pesticide and Toxic Substances (OPTS) Reading Room from 8 a.m. to 4 p.m., Monday through Friday, except legal holidays, in Rm. E–107, 401 M St., SW., Washington, D.C. 20460. The Agency will supplement the record periodically with additional relevant information received.

(Sec. 4, 90 Stat. 2003; (15 U.S.C. 2601))

Dated: December 21, 1983.

William D. Ruckelshaus,

Administrator.

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